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Neonatal diarrhea in llamas and alpacas☆

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Abstract

Diarrhea is an important cause of morbidity in neonatal llamas and alpacas. Diarrhea may be multifactorial in etiology including management and nutritional factors as well as a variety of pathogens. Most of the pathogens involved affect other livestock species and some have host-adapted strains. However, the clinical signs, their expected severity and age of onset of disease varies between species in some cases. The most common pathogens causing diarrhea in neonatal camelids are coronavirus, *Escherichia coli* (*E. coli*), *Cryptosporidium* spp., *Giardia* spp. and coccidia. The purpose of this paper is to review the available literature on neonatal diarrhea in camelids and to present clinical data from 55 cases seen at The Ohio State University.

Keywords: Diarrhea; Cria; Llama; Alpaca; Neonate

1. Introduction

Diarrhea is an important disease in neonatal llamas (*Lama glama)* and alpacas (*Lama pacos).* One study found that diarrhea was the most common cause of morbidity in the pre-weaning period, affecting some 23% of crias (Sharpe et al., unpublished data, 2000). This study monitored 250 crias on four farms over a 5-year period. Commercialization of alpaca breeding in the North America, Australia and Europe, for fiber production has led to increased stocking densities inevitably exposing young stock to higher concentrations of potential pathogens. Nutritional factors, such as overfeeding of bottle-fed orphans, may play a role but infectious pathogens are an increasingly important factor in neonatal diarrhea. A knowledge of the types of agents involved and at what age these infectious agents are likely to cause disease is important in deciding which diagnostic tests to perform and in initiating treatment. Additionally, the clinician must realize that failure to reach a diagnosis and treat the cause of diarrhea effectively will often lead to chronic diarrhea which may ultimately result in chronic renal failure. Failure to receive sufficient immunoglobulins from colostrum during the first 12 h of life predisposes the neonate to bacterial, viral and protozoal agents but each tends to have a characteristic clinical presentation and this may help guide the clinician with regard to

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Table 1
Pathogen isolated from crias with diarrhea in two different studies

Pathogen	Cebra et al. (2003) ($N = 45$)		Data from The Ohio State University (1999–2004) ($N = 58$)[a]	
	Cases (%)	Age range (in days)	Cases (%)	Age range (in days)
E. coli	–	–	0	–
Rotavirus	2	210	0	–
Coronavirus	42	10–150	6.9	9–94
Cryptosporidium spp	9	10–45	25.9	7–100
Giardia	18	10–120	32.8	7–120
Coccidia	13	21–60	12.1	21–104
Salmonella	0	–	1.7	45
Nematode ova	2	–	1.7	80
Undetermined cause	–	–	36.2	4–80

[a] This is retrospective data from clinical cases of diarrhea in crias aged less than 4 months at time of diagnosis.

therapeutic options. This review of the current literature regarding diarrhea in camelid neonates will include clinical experience of neonatal diarrhoea seen at The Ohio State University Veterinary Teaching Hospital between 1999 and 2004.

The main causes of diarrhea in llama and alpaca crias are listed in Table 1, including the ages at which they are likely to be diagnosed. Differential diagnoses to consider include coccidiosis, *Escherichia coli* (*E. coli*) diarrhea, rotavirus, coronavirus, giardiasis, cryptosporidiosis, salmonellosis, septicemia, nutritional, nematodiasis, tapeworm infestation and metabolic disturbances (e.g. portosystemic shunt).

2. Coccidiosis

Coccidiosis is most commonly diagnosed in neonates and juveniles. Adults are more resistant to clinical disease because of mature immune systems and prior exposure. Coccidiosis is typically associated with overcrowding and poor hygiene. The pathogenesis and severity of the clinical signs observed may be associated with the number of coccidia ingested. Re-infections from a contaminated environment can cause an excessive coccidia burden that may be fatal (Fowler, 1998). Following ingestion of sporulated oocysts, motile stages called sporozoites are released which penetrate epithelial cells in the small intestine. The sporozoites then undergo both sexual and asexual reproductive stages producing oocysts which are shed in the feces. The oocysts cause direct damage to the epithelial mucosa of the small intestine resulting in enteritis and diarrhea. Tenesmus may be observed. Diarrhea may be hemorrhagic and contain shreds of sloughed mucosa. As a result, nutrient malabsorption and subsequent poor growth ensue. Cheney and Allen (1989) report that since the lesions are primarily in the small intestine, fresh blood is rarely seen in affected llamas.

Coccidiosis in camelids is caused by several different species belonging to the genus *Eimeria.* Coccidia tend to be highly species-specific: five species have been identified that affect South American Camelids (SACs); *Eimeria lamae*, *E. alpacae*, *E. macusaniensis*, *E. punoensis* and *E. peruviana* (Fowler, 1998; Guerrero, 1967; Guerrero et al., 1971; Schrey et al., 1991). All five species have been detected in fecal samples from camelids in the USA (Fowler, 1998). A distinguishing feature of the *E. macusaniensis* oocyst is that it is much larger than the other four species, measuring 81–107 μm in length, and has a very thick wall (Guerrero et al., 1971). In contrast, the other four species measure 17–40 μm in length and have a thin-walled oocyst (Guerrero, 1967).

The life cycle is not particularly well-established for coccidia specific to SACs. However, Foreyt (2001) lists prepatent periods for four out of the five species that affect South American Camelids. These vary from as little as 10 days for *E. punoensis* to 33–34 days for *E. macusaniensis*. The prepatent periods for *E. alpacae* and *E. punoensis* were established from experimental data using fecal samples from four llamas (Foreyt and Lagerquist, 1992). The authors have seen clini-

cal coccidiosis in alpacas as early as 21 days of age. Identification of the particular species involved in this case was not performed but such an early appearance of clinical disease suggests that neonates exposed to contaminated environments can become infected in the first few days of life.

Rosadio and Ameghino (1994) described an outbreak of coccidiosis on a farm in southern Peru that was suspected to have caused the deaths of 12 alpacas between 25 and 35 days of age. Eight of these cases had been observed with pre-mortem diarrhea, four others had died suddenly. At necropsy, all of the carcasses were observed to be thin and dehydrated. Gross examination revealed gas-distended intestines with reddened intestinal mucosa throughout the small intestine and the mesenteric lymph nodes were enlarged. Histopathologically, there was partial ulceration and moderate necrosis of the small intestinal mucosa with shortened intestinal villi. Throughout the mucosa there were large numbers of sexual and asexual stages of a coccidian that was thought to be *E. macusaniensis.* These were causing marked disruption of the epithelial crypt glands. Cebra et al. (2003) isolated *Eimeria* spp. 6 of 45 (13%) unweaned llama or alpaca crias with diarrhea. The six crias were between 21 and 60 days old. The peak times for diarrhea case submissions corresponded with the wetter months of Fall and Winter and at these times, coccidia were isolated more frequently. A case of coccidiosis and leptospirosis was reported in a 3-month-old guanaco (Hodgin et al., 1984). In this particular case, the cria died without pre- or post-mortem evidence of diarrhea and lesions of subacute enteritis with associated coccidian reproductive stages were confined to a relatively small portion (60 cm) of jejunum. The coccidiosis was not thought to have contributed to the death of this cria. Costarella and Anderson (1999) reported a case of ileocecocolic intussusception in a 1-month-old llama cria. A fecal examination had revealed many coccidial oocysts but there had been no signs of diarrhea and a causal relationship was considered but could not be established.

Several other reports have documented the occurrence of coccidial oocysts in feces from healthy SACs in the United States (Jarvinen, 1999; Rickard and Bishop, 1988; Schrey et al., 1991). All these studies involved large sample sizes of 443, 239 and 144 animals respectively. In two of the studies, animals less than 1-year-old had the highest prevalence of oocyst shedding (Jarvinen, 1999; Rickard and Bishop, 1988). Information on age of animals was not available in the study by Schrey et al. (1991). Method of fecal examination can affect the yield of oocysts detected (Jarvinen, 1999). Flotation solutions with a specific gravity of $\leq$1.20 (i.e. saturated sodium chloride which is 1.20) may fail to allow detection of *E. macusaniensis* oocysts which are larger and heavier. In comparison, a sugar solution with specific gravity of 1.28–1.30 (used at a 1:10 dilution for oocyst detection by centrifugal flotation) gave more reliable results, especially when low concentrations of oocysts were present.

Effective treatment of clinical coccidiosis is achieved using oral sulfadimethoxine (Albon, Pfizer, USA) at a dose of 15 mg/kg twice daily for 5 days. Amprolium may also be used orally at 10 mg/kg once daily for 5 days (Smith, 1999). Amprolium inhibits differentiation of merozoites by acting on first generation schizonts in the small intestine (Bennett et al., 2001). It does not kill the merozoites: these enter the sexual reproductive phase which ultimately produces oocysts. Therefore, an animal treated with amprolium will still excrete viable oocysts. Clinically affected animals should be isolated and treated. Unaffected animals from the same pen should also be treated since they will have been exposed and may be harboring susceptible coccidian stages.

Good management practices and maintenance of hygienic facilities for young animals should be considered the most important factors in prevention of coccidiosis. Strategic use of anticoccidial drugs may also be considered as a supplemental means of controlling coccidiosis and should be used on a herd basis. Since outbreaks of coccidiosis are common in the wetter months, prophylactic drug regimes should be used during these times of year. Consideration should be given to application of preventative measures before and during stressful events such as weaning, shearing or herd movements. Amprolium may be used prophylactically and is added to water at 5 mg/kg body weight daily over a 21-day period. Correct dosage of amprolium is important since overdosing causes thiamine deficiency and can produce clinical polioencephalomalacia. Alternatively, decoquinate may be used more safely added into feed at 0.5 mg/kg/day for 28 days. Ionophore antibiotics such as monensin or salinomycin are toxic for camelids and should not be used to prevent coccidiosis in these species.

3. *E. coli* diarrhea

E. coli diarrhea often occurs in combination with neonatal septicemia. Affected neonates, typically 3–7 days old, are severely ill with profuse, watery diarrhea, lethargy, dehydration and may have abdominal distension. Colibacillosis occurs in neonates secondary to failure of passive transfer of maternal immunity in neonates but can occur secondary to other gastrointestinal diseases such as viral enteritis in slightly older animals. If *E. coli* septicemia and diarrhea is not treated intensively early on, rapid progression of clinical signs may occur. Water and electrolyte losses can be severe, with bicarbonate loss being particularly significant.

The enterotoxigenic *E. coli* (ETEC) strains are a leading cause of neonatal loss in livestock but have not been specifically identified in llamas and alpacas. ETEC strains of *E. coli* bacteria possess adhesins, or surface proteins, called fimbriae which allow attachment of the bacteria to mucosal cells in the small intestine, predominantly the distal jejunum and proximal ileum (DebRoy and Maddox, 2001). Following attachment, the bacteria then produce either heat-labile (LT) or heat-stable (ST) toxins resulting in increased secretion of water and electrolytes by the intestinal cells (hypersecretory diarrhea). Villous atrophy and sloughing of enterocytes may occur due to hypovolemia, ischemia and circulatory problems in the host—rather than being a specific effect of the bacterial toxins themselves. If *E. coli* infection occurs concurrently with rotavirus or coronavirus infection, then the villous atrophy may be extensive and severe. Different combinations of virulence factors (fimbriae and enterotoxins) determine the classification of ETEC strains, the majority of which are plasmid-encoded. Commonly-known fimbriae are K88, K99 and F41 while STa is the enterotoxin found in cattle isolates. It is not known specifically which virulence factors might be produced by ETEC affecting camelid species although the pathogenesis of the disease is presumed to be the same.

Various diagnostic tests have been done in other species detecting virulence factors expressed by ETEC strains. These include DNA probes and polymerase chain reaction (PCR). In addition, latex agglutination and ELISA kits are now available for detecting the most common virulence factors involved in neonatal diarrhea (K88, K99, F41 and 987P). ETEC rapidly lose fimbriae in microbial culture (or in vitro) and are difficult to differentiate from common enteric *E. coli*. Although enteropathogenic *E. coli* has been described in cattle, the authors know of no cases reported in llamas or alpacas.

Gram-negative bacterial infections were reported in five neonatal llamas and one alpaca (Adams and Garry, 1992). All were aged 1–5 days and had positive blood cultures. Three of the crias presented with diarrhea and all three died soon after admission to the hospital. *E. coli* was cultured from three of the six cases, though it is not clear whether these were the crias that presented with diarrhea. However, all three of the crias that had diarrhea were necropsied and had lesions of hemorrhagic enteritis evident grossly. Bacterial culture did not yield any *Clostridium* sp. in these cases: assays for clostridial toxins were not performed. Diagnostic tests for viral pathogens or parasites were not done.

The authors' experience, suspected clinical cases of *E. coli* diarrhea are often leukopenic with a degenerative left shift neutrophilia. Acutely ill young crias (<7 days old) with diarrhea and these characteristic findings on complete blood count should be given broad-spectrum antibiotic with good gram-negative coverage until blood-culture results are available. A typical therapeutic regimen would include potassium penicillin (22,000 IU/kg body weight intravenously every 6 h), gentamicin (5 mg/kg i.v. once daily for 5 days) and supportive intravenous fluid therapy as required. Sick camelid neonates are often hypernatremic and low sodium intravenous fluids are recommended such as 0.45% sodium chloride with 2.5% dextrose.

4. Cryptosporidiosis

Cryptosporidium species are zoonotic protozoan pathogens and can cause severe and sometimes fatal outbreaks of diarrhea in neonatal animals as well as immunocompromised individuals. All mammals appear to be susceptible. *Cryptosporidium* species lack host specificity. Numerous species have been recognized including species affecting birds and reptiles but *Cryptosporidium parvum* has been investigated most extensively and is believed to be responsible for most

mammalian disease (Tzipori and Ward, 2002). *Cryptosporidium* spp. were isolated in 9% of cases in one study involving 45 unweaned llama and alpaca crias with diarrhea (Cebra et al., 2003).

Infection occurs by fecal–oral route and results from the ingestion of contaminated food or water. No intermediate host is required. *Cryptosporidium* oocysts are extremely resistant to decay in the environment and to most disinfectants. As few as 10 oocysts can induce disease in susceptible individuals while clinically affected calves can shed greater than 10^{10} oocysts during infections forming a significant source of infection for other animals (Tzipori and Ward, 2002). Additionally, asymptomatic adult cattle are thought to be a major source of environmental contamination. The exact mechanism by which *Cryptosporidium* attaches and invades enterocytes is poorly understood at the present time. However, it is known that following attachment, *Cryptosporidium* forms a parasitophorous vacuole within the host cell (intracellular but extracytoplasmic) bounded by a parasitophorous vacuolar membrane derived from the host cell membrane. This protects the organism from the hostile environment of the gut lumen as well as the host cell's defenses. An additional "feeder organelle membrane" in *Cryptosporidium*, as opposed to other coccidian parasites, is thought to be primarily responsible for nutrient uptake from the host cell. Inclusion of *Cryptosporidium* within enterocytes reduces gastrointestinal absorption in affected individuals due to loss of epithelium, villous atrophy and crypt hyperplasia leading to dehydration and electrolyte imbalance (malabsorptive diarrhea). Maldigestion also occurs as a result of the loss of membrane-bound digestive enzymes such that weight loss and emaciation in neonates is typical. Autoinfection can occur. The authors have diagnosed renal failure in multiple crias with cryptosporidiosis. The mechanism of kidney damage is presumed to be prolonged states of subclinical dehydration leading to reduced renal perfusion and tubular necrosis.

The simplest method of diagnosing cryptosporidiosis is by examination of fecal smears using modified acid-fast stains (Chen et al., 2002; Tzipori and Ward, 2002). Detection of oocysts can be improved by the use of immunofluorescence microscopy and ELISAs. Recently, ELISA tests for both *Cryptosporidium* and *Giardia* oocyst antigens have improved sensitivity and can be easily applied in practice situations. PCR allows speciation and the possibility of identifying the potential source of infection.

Effective treatment for cryptosporidiosis remains elusive. The mainstay of therapy involves supportive care with intravenous fluids and/or total parenteral nutrition due to the malabsorption and maldigestion which occurs in this disease. Lasalocid (0.45 mg/kg q24 for 3 days) has been used by the authors in llamas and alpacas with some success. However, this drug must be dosed carefully to avoid ionophore toxicosis and its efficacy needs further invesitigation. Paromomycin is an aminoglycoside antibiotic that has been used in human cases but studies appear to disagree regarding its efficacy (Tzipori and Ward, 2002). One study showed that it was no more effective than placebo for treating cryptosporidiosis in patients with advanced HIV infection (Hewitt et al., 2000). Several studies have been performed in animals but the results are inconsistent. In one trial, prophylactic paromomycin administration did not reduce the incidence of disease in treated calves but appeared to increase the pre-patent period (Grinberg et al., 2002). A field trial conducted in infected lambs showed significant reduction in excretion of oocysts in lambs treated with paromomycin (100 or 200 mg/kg once daily for 3 consecutive days) as well as a reduction in clinical symptoms (Viu et al., 2000). Other drugs have been tested in humans including azithromycin, sometimes in combination with paromomycin, and nitazoxanide. In Europe, halofuginone has been used with success for prevention of cryptosporidiosis (Hunt, 1999).

The first case of cryptosporidiosis in camelids was reported in an 8-day-old llama (Hovda et al., 1990). The cria developed profuse, watery diarrhea 4 days following surgery to correct a patent urachus. *Cryptosporidium* sp. was isolated from the feces. Tests for salmonella, *Campylobacter*, parasites, rotavirus and coronavirus were negative. The cria deteriorated on initial management consisting of intravenous fluid therapy and ampicillin. After total parenteral nutrition was initiated, the diarrhea resolved within 3 days and the cria made a steady recovery. In this case, stress of anesthesia and surgery possibly combined with altered gastrointestinal microflora due to the use of oral antibiotics (trimethoprim-sulfadiazine) were thought to have resulted in clinical cryptosporidiosis. The authors considered that other pathogens such as viruses or bacteria

may have been involved in the development of diarrhea but none could be documented.

Three fatal cases of cryptosporidiosis were reported in alpaca crias aged between 9 and 30 days old at time of death (Bidewell and Cattell, 1998). The 9-day-old cria had yellow diarrhea and died within 12 h; the 12-day-old cria had no diarrhea but died within 24 h of being found in a moribund state and the 30-day-old cria had a history of diarrhea and weight loss occurring over 7 days prior to death. At necropsy, small and large intestinal congestion were noted, the severity of which varied by case. All three had distended large intestines whereas only the two older cases also had small intestinal distension. Large numbers of cryptosporidia oocysts consistent with *Cryptosporidium parvum* were identified in smears of intestinal content using a modified Ziehl–Neelson stain. In addition, fecal analysis yielded no significant organisms or coccidia, an ELISA for clostridial toxins (α, β and ε) was negative and electron microscopy was negative for viruses.

Gracenea et al. (2002) investigated the transmission dynamics of *Cryptosporidium* in various primate and herbivore species at the Barcelona zoo. This study included two dromedary and four bactrian camels. Fecal samples were collected monthly for 3 consecutive days over a period of 1 year and examined for *Cryptosporidium* oocysts. All camels in the study were found to shed *Cryptosporidium* oocysts despite being asymptomatic. Only one bactrian shed year-round. The other camels had re-infections throughout the year with negative periods of more than 3 consecutive months between periods of shedding. There was a statistically significant effect of season on oocyst shedding with the highest levels occurring in autumn and winter when the animals were confined to smaller areas indoors. Fecal shedding of *Cryptosporidium* oocysts has not been demonstrated in asymptomatic SACs. No *Cryptosporidium* oocysts were found in a study on 354 llamas aged between 3 weeks and 23 years (Rulofson et al., 2001).

5. Giardia

Giardia infection primarily occurs from contaminated water sources and is a zoonotic disease. The oocysts can survive about 3 months in water at 4 °C. Since the organism affects the small intestine where it causes villous atrophy, a malabsorptive diarrhea results in dehydration and weight loss.

Giardia was reported for the first time in a llama in 1987 (Kiorpes et al., 1987). The llama was several months old and asymptomatic at the time of fecal examination. Giardia was the pathogen responsible for 18% of 45 cases of diarrhea in unweaned llama and alpaca crias (Cebra et al., 2003). Fecal shedding of *Giardia duodenalis* was reported in 3.4% of 354 asymptomatic llamas with crias being significantly more likely to shed oocysts than older llamas (Rulofson et al., 2001). The risk factors for increased oocyst shedding included having more than 10 yearlings on the property, smaller pen sizes and large unit sizes of more than 20 animals. In the authors' experience, oral fenbendazole at 50 mg/kg once daily for 5 consecutive days is effective at treating giardiasis in llamas and alpacas.

6. Salmonellosis

Salmonella spp. do not appear to be a common cause of diarrhea in llamas or alpacas. *Salmonella* was not isolated in any of 45 cases of cria diarrhea in one study (Cebra et al., 2003). A study on 76 healthy llamas tested for *Salmonella* shedding did not result in any positive case (Rulofson et al., 2001). A field survey on 99 asymptomatic llamas and alpacas residing in Ohio did not result in any positive fecal culture for *Salmonella* (Anderson, D.E., unpublished data, 1999). However, septicemic salmonellosis was reported in one 6-year-old female llama and one 6-day-old llama cria (Anderson et al., 1995). *S. typhimurium* was isolated from blood culture in the cria. Both cases were fatal but neither displayed any signs of diarrhea. Salmonellosis in the adult llama was attributed to respiratory infection with *S. choleraesuis* var *kunzendorf* which was cultured from the lung tissue. This pathogen is host-adapted to swine and the llama had had fence-line contact with a swine farm.

The authors have isolated various *Salmonella* species (*S. ohio, S. newport, S. choleraesuis*) from young (<6-month-old) llamas and alpacas with diarrhea. These animals presented with severe, protracted diarrhea and dehydration. Several cases had clinical signs of septicemia (scleral injection, tachycardia, tachypnoea, elevated rectal temperature) and were positive on blood culture for *Salmonella* species. A

degenerative left shift neutrophilia was present on hematology.

7. Viral diarrhea

Both rotavirus and coronavirus have been identified as causing diarrhea in neonatal llamas and alpacas. Coronavirus was the most common pathogen causing diarrhea in unweaned crias in one recent study (Cebra et al., 2003). It was identified by electron microscopy in 42% of cases and affected 64% of herds studied with an age distribution from 10–150 days at the time of diagnosis. In the same study, this was the only pathogen involved in outbreaks that also affected adults. Coronavirus also was found to cause diarrhea at any time of the year in contrast to coccidiosis which was more prevalent in wetter times of year (Fall and Spring). In contrast, rotavirus was found in only 2% (1/45) of cases.

Parreño et al. (2001) investigated the presence of rotavirus and coronavirus in two farms of captured guanacos in Patagonia (Argentina). Both farms suffered severe diarrhea outbreaks with a morbidity rate of 100% and a mortality rate of 83%. Animals were between 1 day and 4 months of age at time of capture, kept in small yards and raised on powdered cow's milk. In all cases, disease was acute in onset and the feces were dark-green. Affected animals were 7–40 days old. Death occurred within 2–6 days after dehydration. Rotavirus was detected in the feces of two neonates (aged 2 and 7 days). Analysis of 52 fecal samples from healthy or convalescent animals for rotavirus antigen taken about 30 days after the peak of the outbreak were all negative; however, all animals except the two neonates were positive for rotavirus antibody by ELISA. This included the healthy animals giving total herd prevalences of 95% in both farms. The same animals were all negative for bovine coronavirus antigen in feces and antibody in serum by ELISA.

Puntel et al. (1999) conducted a serological survey for a variety of viruses including bovine rotavirus on 390 llamas in 9 Argentinean farms. Antibody prevalence for rotavirus was 88% and involved all farms tested. The authors surmised that these findings suggest a high susceptibility of llamas to bovine rotavirus. No clinical cases of diarrhea were reported on the farms of study.

Mattson (1994) reported detection of coronavirus by electron microscopy in 2 young llamas, aged 12 days and 9 months, presented with diarrhea.

Treatment of viral diarrhea is mainly supportive. The clinician must rule out and if necessary treat for other pathogens which may be involved concurrently. Various monoclonal antibody vaccines against viral pathogens are available for oral administration to calves and lambs. These may be used safely in camelids on farms experiencing outbreaks of viral diarrhea but they are of unknown efficacy in these species.

8. Nematodiasis

Nematode parasites have been known to cause diarrhea in llamas and alpacas as young as 2 months of age. Young animals are especially susceptible to parasitism since they do not have any acquired resistance. Clinical signs may include ill-thrift, inappetance, anorexia, emaciation in severe cases and diarrhea (Fowler, 1998). The modified Stoll's test (sugar centrifugation test) is the best diagnostic test for identifying gastrointestinal parasites in camelids and utilizes a sugar flotation in combination with centrifugation. In the authors' experience, a 5-day course of oral fenbendazole at 20 mg/kg is effective therapy for nematode-induced diarrhea and other clinical manifestations of this disease.

9. Clostridial enterotoxemia

The authors have cultured beta toxin positive strains of *Clostridium perfringens* type A from several llamas and alpacas with diarrhea. The significance of these findings is unclear. Beta toxin positive strains of *C. perfringens* are increasingly recognized in cattle and may represent an emerging disease. Unlike the clinical syndrome in cattle, many suspected clinical cases in llamas and alpacas have occurred in young stock less than 6 months old and hemorrhage has not been seen in most cases. Evidence of neurotoxin has been seen in some cases displaying head and neck tremors and mild ataxia.

10. Other causes of diarrhea

There has been one reported case in the literature of portosystemic shunt in an alpaca cria (Ivany et al.,

2002). This cria had had recurrent episodes of diarrhea up until the time of diagnosis at 5 months of age. The authors postulated that the diarrhea in portosystemic shunt may be due to "abnormal hepatic metabolism of nutrients or abnormal bacterial growth in the colon".

Nutritional causes of diarrhea in young llamas and alpacas include sudden access to lush Spring pastures, grain overload or overfeeding milk to bottle-fed crias. The authors have seen crias develop diarrhea when bottle-fed with milk replacers or goats' milk at rates greater than 15% of their body weight on a daily basis. Cebra et al. (1996) reported on six cases of forestomach acidosis including one llama that was 2 months old. The crias from that group were creep-fed a grain mixture of equal parts of rolled barley, rolled oats and cracked corn.

Antibiotic-induced diarrhea should be considered a differential diagnosis in crias with a history of antibiotic treatment, especially if orally administered.

11. Conclusions

In conclusion, llama and alpaca crias are susceptible to diarrhea caused by a similar range of pathogens to those affecting other domestic species. In crias aged less than 7 days, diarrhea is most likely to be caused either by nutritional factors in bottle-fed neonates or by gram negative infections, especially when colostrum ingestion has been inadequate. In the latter situation, the diarrhea is likely to be accompanied by septicemia and affected crias will be quite ill. Viral diarrhea rarely affects crias younger than 7 days and in the US is mostly due to coronavirus. Cryptosporidium and giardia also affect crias aged 7 days or older and these pathogens tend to be more of a problem on large farms. However, the common practice of moving females with their crias during the neonatal period for breeding purposes with inadequate quarantine provision may result in infections on small units also. In addition, giardia may be a problem on farms using surface ponds as their water supply. Coccidiosis does not seem to occur in crias younger than 3 weeks but must be addressed as a herd issue when it is recognized. Diarrhea caused by gastrointestinal parasites is unlikely in crias younger than 2 months. It is important to recognize that multiple pathogen disease occurs, especially with cryptosporidium and this may affect treatment options and rate of recovery. Diagnostic tests are available for all of these pathogens and appropriate testing will aid the clinician in directing treatment for the individual and in helping to control the spread of disease among susceptible animals in the herd.

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